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NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable

NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/CAplus

NEWS 22 FEB 05 German (DE) application and patent publication number format changes

NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 11:28:33 ON 01 MAR 2004

=> file registry

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 11:28:42 ON 01 MAR 2004

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STRUCTURE FILE UPDATES: 29 FEB 2004 HIGHEST RN 656221-41-9

DICTIONARY FILE UPDATES: 29 FEB 2004 HIGHEST RN 656221-41-9

TSCA INFORMATION NOW CURRENT THROUGH JULY 14, 2003

Please note that search-term pricing does apply when conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more information enter HELP PROP at an arrow prompt in the file or refer to the file summary sheet on the web at:
<http://www.cas.org/ONLINE/DBSS/registryss.html>

=> s wormannin/cn

L1 0 WORMANNIN/CN

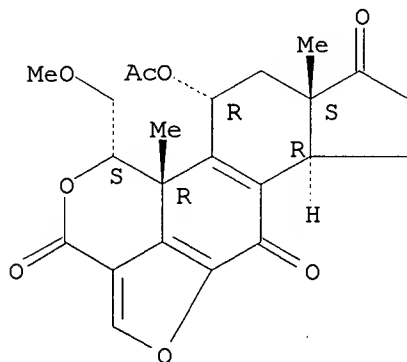
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=> s wortmannin/cn
L2          1 WORTMANNIN/CN

=> d 11
L1 HAS NO ANSWERS
L1          0 SEA FILE=REGISTRY ABB=ON  WORMANNIN/CN
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=> d 12
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L2  ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2004 ACS on STN
RN  19545-26-7  REGISTRY
CN  3H-Furo[4,3,2-de]indeno[4,5-h]-2-benzopyran-3,6,9-trione,
    11-(acetyloxy)-1,6b,7,8,9a,10,11,11b-octahydro-1-(methoxymethyl)-9a,11b-
    dimethyl-, (1S,6bR,9aS,11R,11bR)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN  3H-Furo[4,3,2-de]indeno[4,5-h]-2-benzopyran-3,6,9-trione,
    11-(acetyloxy)-1,6b,7,8,9a,10,11,11b-octahydro-1-(methoxymethyl)-9a,11b-
    dimethyl-, [1S-(1.alpha.,6b.alpha.,9a.beta.,11.alpha.,11b.beta.)]-
OTHER NAMES:
CN  KY 12420
CN  Wortmannin
FS  STEREOSEARCH
DR  1405-03-4
MF  C23 H24 O8
CI  COM
LC  STN Files:  ADISINSIGHT, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS,
    BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CHEMCATS, CIN,
    CSChem, DDFU, DRUGU, EMBASE, IPA, MEDLINE, MRCK*, NAPRALERT, PROMT,
    RTECS*, TOXCENTER, USPAT2, USPATFULL
    (*File contains numerically searchable property data)
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Absolute stereochemistry.



****PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT****

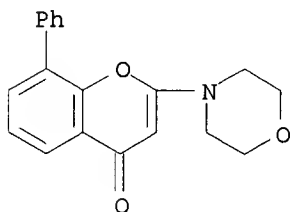
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482 REFERENCES IN FILE CA (1907 TO DATE)
  8 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
482 REFERENCES IN FILE CAPLUS (1907 TO DATE)
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=> s LY294002/cn
L3          0 LY294002/CN
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=> s LY 294002/cn
L4 1 LY 294002/CN

=> d 14

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2004 ACS on STN
RN 154447-36-6 REGISTRY
CN 4H-1-Benzopyran-4-one, 2-(4-morpholinyl)-8-phenyl- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 8-Phenyl-2-(morpholin-4-yl)-chromen-4-one
CN **LY 294002**
CN NSC 697286
FS 3D CONCORD
MF C19 H17 N O3
SR CA
LC STN Files: ADISINSIGHT, AGRICOLA, BIOSIS, BIOTECHNO, CA, CANCERLIT,
CAPLUS, CHEMCATS, CSCHEM, EMBASE, MEDLINE, TOXCENTER, USPAT2, USPATFULL



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

187 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
188 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> file uspatfull
COST IN U.S. DOLLARS
FULL ESTIMATED COST

SINCE FILE	TOTAL
ENTRY	SESSION
22.94	23.15

FILE 'USPATFULL' ENTERED AT 11:31:10 ON 01 MAR 2004
CA INDEXING COPYRIGHT (C) 2004 AMERICAN CHEMICAL SOCIETY (ACS)

FILE COVERS 1971 TO PATENT PUBLICATION DATE: 26 Feb 2004 (20040226/PD)
FILE LAST UPDATED: 26 Feb 2004 (20040226/ED)
HIGHEST GRANTED PATENT NUMBER: US6698023
HIGHEST APPLICATION PUBLICATION NUMBER: US2004040063
CA INDEXING IS CURRENT THROUGH 26 Feb 2004 (20040226/UPCA)
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 26 Feb 2004 (20040226/PD)
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Dec 2003
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Dec 2003

>>> USPAT2 is now available. USPATFULL contains full text of the	<<<
>>> original, i.e., the earliest published granted patents or	<<<
>>> applications. USPAT2 contains full text of the latest US	<<<
>>> publications, starting in 2001, for the inventions covered in	<<<
>>> USPATFULL. A USPATFULL record contains not only the original	<<<
>>> published document but also a list of any subsequent	<<<
>>> publications. The publication number, patent kind code, and	<<<

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>>> publication date for all the US publications for an invention <<<
>>> are displayed in the PI (Patent Information) field of USPATFULL <<<
>>> records and may be searched in standard search fields, e.g., /PN, <<<
>>> /PK, etc. <<<

>>> USPATFULL and USPAT2 can be accessed and searched together <<<
>>> through the new cluster USPATALL. Type FILE USPATALL to <<<
>>> enter this cluster. <<<
>>> <<<
>>> Use USPATALL when searching terms such as patent assignees, <<<
>>> classifications, or claims, that may potentially change from <<<
>>> the earliest to the latest publication. <<<

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This file contains CAS Registry Numbers for easy and accurate substance identification.

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=> s 19545-26-7 RN
      2 19545-26-7
      10708 RN
L5      0 19545-26-7 RN
          (19545-26-7(W)RN)

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=> s 19545-26-7/RN
L6      54 19545-26-7/RN

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=> s l6 and psoriasis
      17597 PSORIASIS
L7      11 L6 AND PSORIASIS

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=> s l7 and pd<1999
      2436404 PD<1999
          (PD<19990000)
L8      0 L7 AND PD<1999

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=> d l7 1-11

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L7 ANSWER 1 OF 11 USPATFULL on STN
AN 2003:273414 USPATFULL
TI Methods for modulating T cell responses by manipulating intracellular
   signal transduction
IN June, Carl H., Rockville, MD, United States
PA The United States of America as represented by the Secretary of the
   Navy, Washington, DC, United States (U.S. government)
PI US 6632789 B1 20031014
AI US 1994-245282 19940429 (8)
DT Utility
FS GRANTED
LN.CNT 1110
INCL INCLM: 514/001.000
     INCLS: 514/453.000; 424/130.100; 424/278.100
NCL NCLM: 514/001.000
     NCLS: 424/130.100; 424/278.100; 514/453.000
IC [7]
     ICM: A01N061-00
     ICS: A01N043-16; A61K039-395; A61K045-00
EXF 435/240.2; 435/240.1; 435/244; 424/278.1; 424/130.1; 514/453; 514/1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

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L7 ANSWER 2 OF 11 USPATFULL on STN
AN 2003:173938 USPATFULL
TI Combinations for the treatment of immunoinflammatory disorders
IN Keith, Curtis, Boston, MA, UNITED STATES

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Borisys, Alexis, Boston, MA, UNITED STATES
Zimmerman, Grant, Somerville, MA, UNITED STATES
Jost-Price, Edward Roydon, West Roxbury, MA, UNITED STATES
Manivasakam, Palaniyandi, Brighton, MA, UNITED STATES
Hurst, Nicole, Boston, MA, UNITED STATES
Foley, Michael A., Chestnut Hill, MA, UNITED STATES

PI US 2003119786 A1 20030626
AI US 2002-264991 A1 20021004 (10)
PRAI US 2001-327674P 20011005 (60)

DT Utility
FS APPLICATION

LN.CNT 1182

INCL INCLM: 514/081.000
INCLS: 514/171.000; 514/262.100

NCL NCLM: 514/081.000
NCLS: 514/171.000; 514/262.100

IC [7]
ICM: A61K031-675
ICS: A61K031-56; A61K031-519

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 3 OF 11 USPATFULL on STN

AN 2003:23354 USPATFULL

TI Intravascular delivery of mycophenolic acid
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES

PA Avantec Vascular Corporation, Sunnyvale, CA, UNITED STATES, 94086 (U.S. corporation)

PI US 2003017190 A1 20030123
AI US 2002-242334 A1 20020911 (10)

RLI Division of Ser. No. US 2001-782927, filed on 13 Feb 2001, GRANTED, Pat. No. US 6471980

PRAI US 2000-258024P 20001222 (60)

DT Utility
FS APPLICATION

LN.CNT 1014

INCL INCLM: 424/426.000
INCLS: 514/470.000; 514/171.000; 514/251.000; 514/291.000

NCL NCLM: 424/426.000
NCLS: 514/470.000; 514/171.000; 514/251.000; 514/291.000

IC [7]
ICM: A61K031-573
ICS: A61K031-525; A61K031-4745; A61F002-00; A61K031-365

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 4 OF 11 USPATFULL on STN

AN 2003:23305 USPATFULL

TI Purposeful movement of human migratory cells away from an agent source
IN Poznansky, Mark C., Charlestown, MA, UNITED STATES
Luster, Andrew D., Wellesley, MA, UNITED STATES
Scadden, David T., Weston, MA, UNITED STATES

PI US 2003017141 A1 20030123
AI US 2002-191988 A1 20020709 (10)

RLI Division of Ser. No. US 2000-546153, filed on 7 Apr 2000, GRANTED, Pat. No. US 6448054

PRAI US 1999-128272P 19990408 (60)
US 1999-168952P 19991203 (60)

DT Utility
FS APPLICATION

LN.CNT 2813

INCL INCLM: 424/093.700
INCLS: 435/372.000; 435/366.000

NCL NCLM: 424/093.700
NCLS: 435/372.000; 435/366.000
IC [7]
ICM: A61K045-00
ICS: C12N005-08
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 5 OF 11 USPATFULL on STN
AN 2002:259409 USPATFULL
TI Method for regulating angiogenesis
IN Hla, Timothy, Avon, CT, UNITED STATES
Lee, Meng-Jer, Unionville, CT, UNITED STATES
Claffey, Kevin P., Burlington, CT, UNITED STATES
Ancellin, Nicolas, Farmington, CT, UNITED STATES
Thangada, Shobha, Glastonbury, CT, UNITED STATES
PI US 2002142982 A1 20021003
AI US 2001-945353 A1 20010831 (9)
RLI Continuation-in-part of Ser. No. US 2000-651846, filed on 31 Aug 2000,
PENDING
PRAI US 1999-152266P 19990902 (60)
DT Utility
FS APPLICATION
LN.CNT 1830
INCL INCLM: 514/044.000
INCLS: 514/453.000
NCL NCLM: 514/044.000
NCLS: 514/453.000
IC [7]
ICM: A61K048-00
ICS: A61K031-366
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 6 OF 11 USPATFULL on STN
AN 2002:230824 USPATFULL
TI Purposeful movement of human migratory cells away from an agent source
IN Poznansky, Mark C., Charlestown, MA, United States
Luster, Andrew T., Wellesley, MA, United States
Scadden, David T., Weston, MA, United States
PA The General Hospital Corporation, Boston, MA, United States (U.S.
corporation)
PI US 6448054 B1 20020910
AI US 2000-546153 20000407 (9)
PRAI US 1999-128272P 19990408 (60)
US 1999-168952P 19991203 (60)
DT Utility
FS GRANTED
LN.CNT 2817
INCL INCLM: 435/184.100
INCLS: 424/085.100
NCL NCLM: 424/184.100
NCLS: 424/085.100
IC [7]
ICM: C12N009-99
ICS: A61K045-00
EXF 424/184.1; 424/85.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 7 OF 11 USPATFULL on STN
AN 2002:213450 USPATFULL
TI Intravascular delivery of mycophenolic acid
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES

PI US 2002114823 A1 20020822
US 6471980 B2 20021029
AI US 2001-782927 A1 20010213 (9)
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1135
INCL INCLM: 424/423.000
INCLS: 514/470.000
NCL NCLM: 424/423.000
NCLS: 424/424.000; 424/425.000; 424/426.000
IC [7]
ICM: A61F002-00
ICS: A61K031-365

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 8 OF 11 USPATFULL on STN
AN 2002:158065 USPATFULL
TI Delivery or therapeutic capable agents
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PA AVANTEC VASCULAR CORPORATION, San Jose, CA (U.S. corporation)
PI US 2002082679 A1 20020627
AI US 2001-2595 A1 20011101 (10)
PRAI US 2000-258024P 20001222 (60)
US 2001-308381P 20010726 (60)
DT Utility
FS APPLICATION
LN.CNT 3153
INCL INCLM: 623/001.150
INCLS: 623/001.420; 424/426.000
NCL NCLM: 623/001.150
NCLS: 623/001.420; 424/426.000
IC [7]
ICM: A61F002-06
ICS: A61F002-00

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 9 OF 11 USPATFULL on STN
AN 2002:158064 USPATFULL
TI Intravascular delivery of mizoribine
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PI US 2002082678 A1 20020627
AI US 2001-783254 A1 20010213 (9)
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1050
INCL INCLM: 623/001.150
NCL NCLM: 623/001.150
IC [7]
ICM: A61F002-06

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 10 OF 11 USPATFULL on STN
AN 2002:158063 USPATFULL
TI Intravascular delivery of methylprednisolone
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PI US 2002082677 A1 20020627
AI US 2001-782804 A1 20010213 (9)

PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1074
INCL INCLM: 623/001.150
INCLS: 623/001.430
NCL NCLM: 623/001.150
NCLS: 623/001.430
IC [7]
ICM: A61F002-06
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L7 ANSWER 11 OF 11 USPATFULL on STN
AN 2002:119323 USPATFULL
TI Inhibitors of angiogenesis and tumor growth for local and systemic administration
IN Singh, Saira Sayed, Los Gatos, CA, UNITED STATES
PI US 2002061303 A1 20020523
US 6696483 B2 20040224
AI US 2001-971062 A1 20011003 (9)
PRAI US 2000-237429P 20001003 (60)
DT Utility
FS APPLICATION
LN.CNT 1145
INCL INCLM: 424/094.630
INCLS: 514/183.000; 514/008.000; 514/449.000; 514/559.000; 514/029.000;
514/034.000; 514/171.000; 514/263.300; 514/045.000; 514/050.000;
424/649.000
NCL NCLM: 514/450.000
NCLS: 514/457.000; 514/690.000; 514/725.000
IC [7]
ICM: A61K038-16
ICS: A61K038-48; A61K031-7048; A61K031-704; A61K031-365; A61K031-337
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 17 1-11 ab, kwic

L7 ANSWER 1 OF 11 USPATFULL on STN
AB Methods for modulating T cell responses by manipulating intracellular signals associated with T cell costimulation are disclosed. The methods involve inhibiting or stimulating the production of at least one D3-phosphoinositide in a T cell. Production of D3-phosphoinositides can be manipulated by contacting a T cell with an inhibitor or activator of phosphatidylinositol 3-kinase. Inhibitors of phosphatidylinositol 3-kinase for use in the methods of the invention include wortmannin and quercetin, or derivatives or analogues thereof. The methods of the invention can further comprise modulating other intracellular signals associated with costimulation, such as protein tyrosine phosphorylation, for example by modulating the activity of a protein tyrosine kinase or a protein tyrosine phosphatase in the T cell. Inhibition of a T cell response in accordance with the disclosed methods is useful therapeutically in situations where it is desirable to inhibit an immune response to an antigen(s), for example in organ or bone marrow transplantation and autoimmune diseases. Alternatively, stimulation of a T cell response in accordance with the disclosed methods is useful therapeutically to enhance an immune response to an antigen(s), for example to stimulate an anti-tumor response in a subject with a tumor, to stimulate a response against a pathogenic agent or increase the efficacy of vaccination. Novel screening assays for identifying inhibitors or activators of phosphatidylinositol 3-kinase, which can be used to inhibit or stimulate a T cell response, are also disclosed.

DETD . . . disorders associated with an inappropriate or abnormal immune response include rheumatoid arthritis, juvenile rheumatoid arthritis, psoriatic arthritis, allergies, contact dermatitis, **psoriasis**, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, multiple sclerosis, allergic encephalomyelitis, systemic lupus erythematosus, acute necrotizing hemorrhagic encephalopathy, idiopathic.

IT 117-39-5, Quercetin 19545-26-7, Wortmannin 70563-58-5,
Herbimycin A 154447-36-6, LY 294002
(methods for modulating T-cell responses by manipulating intracellular signal transduction)

L7 ANSWER 2 OF 11 USPATFULL on STN

AB The invention features a method for treating a patient having an immunoinflammatory disorder, by administering to the patient (i) a tetra-substituted pyrimidopyrimidine, and (ii) a corticosteroid simultaneously or within 14 days of each other in amounts sufficient to reduce or inhibit immunoinflammation.

SUMM [0003] Immunoinflammatory disorders (e.g., rheumatoid arthritis, **psoriasis**, ulcerative colitis, Crohn's disease, stroke-induced brain cell death, ankylosing spondylitis, fibromyalgia, and inflammatory dermatoses, asthma, multiple sclerosis, type I diabetes, . . .

SUMM . . . diseases or disorders treated using the methods and compositions of this invention are immunoinflammatory disorders including, for example, rheumatoid arthritis, **psoriasis**, ulcerative colitis, Crohn's disease, stroke-induced brain cell death, ankylosing spondylitis, fibromyalgia, asthma, multiple sclerosis, type I diabetes, systemic lupus erythematosus, . . .

SUMM . . . Immunoinflammatory disorders result in the destruction of healthy tissue by an inflammatory process. Examples of immunoinflammatory disorders include, rheumatoid arthritis, **psoriasis**, ulcerative colitis, Crohn's disease, stroke-induced brain cell death, ankylosing spondylitis, fibromyalgia, asthma, multiple sclerosis, type I diabetes, systemic lupus erythematosus, . . .

CLM What is claimed is:
13. The method of claim 1, wherein said immunoinflammatory disorder is rheumatoid arthritis, **psoriasis**, ulcerative colitis, Crohn's disease, inflammatory dermatosis, or stroke induced brain cell death.

IT 50-02-2, Dexamethasone 50-03-3, Hydrocortisone acetate 50-04-4, Cortisone acetate 50-23-7, Hydrocortisone 50-24-8, Prednisolone 52-21-1, Prednisolone-21-acetate 53-03-2, Prednisone 53-06-5, Cortisone 53-33-8, Paramethasone 53-34-9, Fluprednisolone 53-35-0, 6.beta.-Hydroxycortisol 53-36-1, Methylprednisolone acetate 58-32-2, Dipyrindamole 67-73-2, Fluocinolone acetonide 67-78-7, Triamcinolone diacetate 76-25-5, Triamcinolone acetonide 76-43-7, Fluoxymesterone 79-60-7 83-43-2, Methylprednisolone 124-94-7, Triamcinolone 125-02-0, Prednisolone sodium phosphate 125-04-2, Hydrocortisone sodium succinate 127-31-1, Fludrocortisone 152-58-9, Cortodoxone 338-95-4, Isoflupredone 338-98-7, Isoflupredone acetate 356-12-7, Fluocinonide 357-09-5, Fluorohydroxyandrostenedione 378-44-9, Betamethasone 382-67-2, Desoximethasone 426-13-1, Fluorometholone 508-99-6, Hydrocortisone cypionate 595-52-8, Descinolone 595-77-7, Algestone 599-33-7, Prednylidene 638-94-8, Desonide 641-77-0, 21-Deoxycortisol 1177-87-3, Dexamethasone-21-acetate 1524-88-5, Flurandrenolide 1597-82-6, Paramethasone acetate 1879-77-2, Doxibetasol 2002-29-1, Flumethasone pivalate 2135-17-3, Flumethasone 2152-44-5, Betamethasone-17-valerate 2193-87-5, Fluprednidene 2375-03-3, Methylprednisolone sodium succinate 2557-49-5, Diflorasone 2607-06-9, Diflucortolone 2825-60-7, Formocortal 2920-86-7, Prednisolone-21-hemisuccinate 3093-35-4, Halcinonide 3385-03-3, Flunisolide 3801-06-7, Fluorometholone acetate 3924-70-7, Amcinafal 4419-39-0,

Beclomethasone 4732-48-3, Meclorison 4828-27-7, Clocortolone
 5534-09-8, Beclomethasone dipropionate 5611-51-8, Triamcinolone
 hexacetonide 6000-74-4, Hydrocortisone sodium phosphate 7008-26-6,
 Dichlorison 7681-14-3, Prednisolone tebutate 13609-67-1,
 Hydrocortisone butyrate 13665-88-8, Mopidamol 14484-47-0, Deflazacort
 15001-93-1, Hyrcanoside 17332-61-5, Isoprednidene **19545-26-7**,
 Wortmannin 20423-99-8, Deprodone 21365-49-1, Tralonide 23674-86-4,
 6.alpha.,9.alpha.-Difluoroprednisolone 21-acetate 17-butyrate
 25122-41-2, Clobetasol 25122-46-7, Clobetasol propionate 33564-31-7,
 Diflorasone diacetate 34097-16-0, Clocortolone pivalate 39175-74-1,
 Prednisolone metasulfobenzoate 50629-82-8, Halometasone 51333-22-3,
 Budesonide 54063-32-0, Clobetasone 55879-47-5, 6-Hydroxydexamethasone
 57524-89-7, Hydrocortisone valerate 57781-15-4, Halopredone
 58497-00-0, Procinonide 60135-22-0, Flumoxonide 64096-84-0, NU 3060
 72590-77-3, Hydrocortisone probutate 77011-63-3, Beclomethasone
 dipropionate monohydrate 92626-27-2, Triamcinolone acetonide
 21-palmitate 103638-43-3, Dipyridamole monoacetate 213839-95-3,
 NU3076 256432-41-4, NU 3026 256432-42-5, NU 3059 512165-95-6,
 Prednisolone-21-.beta.-D-glucuronide
 (combinations for treatment of immunoinflammatory disorders)

L7 ANSWER 3 OF 11 USPATFULL on STN

AB The present invention provides improved devices and methods for
 minimizing and/or inhibiting restenosis and hyperplasia after
 intravascular intervention. In particular, the present invention
 provides luminal prostheses which allow for programmed and controlled
 mycophenolic acid delivery with increased efficacy to selected locations
 within a patient's vasculature to inhibit restenosis. An intraluminal
 delivery prosthesis may comprise an expansible structure and means on or
 within the structure for releasing mycophenolic acid at a rate selected
 to inhibit smooth muscle cell proliferation.

DETD [0084] MPA may be combined with other drugs (cytotoxic drugs, cytostatic
 drugs, or **psoriasis** drugs, such as, mizoribine, riboflavin,
 tiazofurin, methylprednisolone, FK 506, zafurin, methotrexate). One drug
 is in or coupled a first coat. . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide
 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2,
 Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone
 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone,
 biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic
 anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6,
 Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol
 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride
 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl
 methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8,
 Cellulose acetate butyrate 9005-49-6, Heparin, biological studies
 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate)
 9016-00-6, Poly(dimethyl siloxane) **19545-26-7**, Wortmannin
 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate
 copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl
 alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone
 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene
 glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol
 methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic
 acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid)
 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1,
 Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol
 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine
 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3,
 Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic
 acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU
 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer

83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin
 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus
 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8,
 Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6,
 Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil
 143653-53-6, Rheopro 152923-56-3, Daclizumab 154447-36-6, LY 294002
 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide
 439112-98-8, Parylant
 (delivery of therapeutic agents)

L7 ANSWER 4 OF 11 USPTAFULL on STN

AB This invention relates to methods and compositions for modulating movement of eukaryotic cells with migratory capacity. More specifically, the invention relates to methods and compositions for modulating movement of cells of hematopoietic, neural, epithelial, or mesenchymal origin, in a specific site in a subject. The foregoing are useful, inter alia, in the treatment of conditions characterized by a need to modulate migratory-cell movement associated with specific sites in a subject. More specifically, specific sites include sites of inflammation and modulation of migratory-cell movement is movement away from an agent source, or repulsion. Other sites include tumor sites, sites of pathogenic infection, and germ cell bearing sites.

SUMM . . . preferred embodiments, the autoimmune disease includes rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's disease, Guillain-Barre syndrome, **psoriasis**, thyroiditis, Graves' disease, myasthenia gravis, glomerulonephritis, autoimmune hepatitis, systemic lupus erythematosus. In further embodiments, the subject has multiple sclerosis, an. . .

DETD . . . as exemplified by diseases such as rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's disease, Guillain-Barre syndrome, **psoriasis**, thyroiditis, Graves' disease, myasthenia gravis, glomerulonephritis, autoimmune hepatitis, multiple sclerosis, systemic lupus erythematosus, etc.

IT 446-72-0, Genistein 19545-26-7, Wortmannin 77699-47-9, Herbimycin
 (cell migration stimulation by polypeptides response to; purposeful movement of human migratory cells away from source of fugetactic agents such as polypeptides)

L7 ANSWER 5 OF 11 USPTAFULL on STN

AB Methods for the inhibition of angiogenesis are presented, comprising affecting the response of the EDG-1 receptor by the administration of pharmaceutically effective antagonists of EDG-1 signal transduction. This invention is based in part on the discovery that Akt protein kinase phosphorylation is required for endothelial cell chemotaxis mediated by the EDG-1 G protein-coupled receptor. This invention relates to the use of modifiers of EDG-1 signal transduction to treat disorders of undesired angiogenesis.

SUMM . . . as solid tumor growth, heart disease, rheumatoid arthritis, peripheral vascular diseases of the elderly, diabetic retinopathy, Kaposi's sarcoma, hemangioma, and **psoriasis**. Angiogenesis is prominent in solid tumor formation and metastasis. Angiogenic factors have been found associated with several solid tumors. A. . .

SUMM . . . of EDG-1 signal transduction is used to treat undesired angiogenesis in tumors, rheumatoid arthritis, diabetic retinopathy, Kaposi's sarcoma, hemangioma or **psoriasis**.

DETD . . . in the neovascularization of tumor cells or other pathological conditions such as rheumatoid arthritis, diabetic retinopathy, Kaposi's sarcoma, hemangioma, and/or **psoriasis**. The oligonucleotides may be adapted or formulated for administration to the body in a number

of ways suitable for the. . .

IT 19545-26-7, Wortmannin 26993-30-6 154447-36-6, LY294002
(EDG-1 signal transduction modifiers for regulating angiogenesis)

L7 ANSWER 6 OF 11 USPATFULL on STN

AB This invention relates to methods and compositions for modulating movement of eukaryotic cells with migratory capacity. More specifically, the invention relates to methods and compositions for modulating movement of cells of hematopoietic, neural, epithelial, or mesenchymal origin, in a specific site in a subject. The foregoing are useful, inter alia, in the treatment of conditions characterized by a need to modulate migratory-cell movement associated with specific sites in a subject. More specifically, specific sites include sites of inflammation and modulation of migratory-cell movement is movement away from an agent source, or repulsion. Other sites include tumor sites, sites of pathogenic infection, and germ cell bearing sites.

SUMM . . . preferred embodiments, the autoimmune disease includes rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's disease, Guillain-Barre syndrome, **psoriasis**, thyroiditis, Graves' disease, myasthenia gravis, glomerulonephritis, autoimmune hepatitis, systemic lupus erythematosus. In further embodiments, the subject has multiple sclerosis, an. . .

DETD . . . as exemplified by diseases such as rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's disease, Guillain-Barre syndrome, **psoriasis**, thyroiditis, Graves' disease, myasthenia gravis, glomerulonephritis, autoimmune hepatitis, multiple sclerosis, systemic lupus erythematosus, etc.

CLM What is claimed is:

. . . 5, wherein the autoimmune disease is rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's disease, Guillain-Barre syndrome, **psoriasis**, thyroiditis, Graves' disease, myasthenia gravis, glomerulonephritis, autoimmune hepatitis, or systemic lupus erythematosus.

IT 446-72-0, Genistein 19545-26-7, Wortmannin 77699-47-9,
Herbimycin
(cell migration stimulation by polypeptides response to; purposeful movement of human migratory cells away from source of fugetactic agents such as polypeptides)

L7 ANSWER 7 OF 11 USPATFULL on STN

AB The present invention provides improved devices and methods for minimizing and/or inhibiting restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for programmed and controlled mycophenolic acid delivery with increased efficacy to selected locations within a patient's vasculature to inhibit restenosis. An intraluminal delivery prosthesis may comprise an expansible structure and means on or within the structure for releasing mycophenolic acid at a rate selected to inhibit smooth muscle cell proliferation.

DETD [0084] MPA may be combined with other drugs (cytotoxix drugs, cytostatic drugs, or **psoriasis** drugs, such as, mizoribine, riboflavin, tiazofurin, methylprednisolone, FK 506, zafurin, methotrexate). One drug is in or coupled a first coat. . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6,

Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) **19545-26-7**, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab 154447-36-6, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylax
 (delivery of therapeutic agents)

L7 ANSWER 8 OF 11 USPATFULL on STN

AB A device and a method using the same, for reducing restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for controlled release of at least one therapeutic capable agent with increased efficacy to selected locations within a patient's vasculature to reduce restenosis. An intraluminal prosthesis may comprise an expandable structure and a source adjacent the expandable structure for releasing the therapeutic capable agent into the body lumen to reduce smooth muscle cell proliferation.

SUMM . . . anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs such as Thalidomide.TM.; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; riboflavin; tiazofurin; zafurin; anti-platelet agents including cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors such as clopidogrel (e.g., Plavix.TM.). . .

DETD . . . amethopterin, is an immunosuppressant and anti-proliferative agent that has been used in the treatment of certain neoplastic diseases and severe **psoriasis**. Chemically Methotrexate.TM. is N-[4[[[(2,4-diamino-6-pteridinyl)methyl] methylamino]benzoyl]-L-glutamic acid. In particular, Methotrexate.TM. is a is inhibits dihydrofolic acid reductase, thereby inhibiting the reduction. . .

DETD . . . anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs such as Thalidomide.TM.; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; riboflavin; tiazofurin; zafurin; anti-platelet agents including cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors such as clopidogrel (e.g., Plavix.TM.). . .

DETD [0180] The therapeutic capable agent may be combined with a second therapeutic capable agent (cytotoxic drugs, cytostatic drugs, or **psoriasis** drugs). One agent is in or coupled to a first coat

while other agent is in or coupled to a. . .

CLM What is claimed is:

- . agents; radiolabelled agents; anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; anti-platelet agents including, cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors ticlopidine phosphodiesterase III inhibitors, glycoprotein IIb/IIIa agents; eptifibatides, . . .
- . agents; radiolabelled agents; anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; anti-platelet agents including, cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors ticlopidine phosphodiesterase III inhibitors, glycoprotein IIb/IIIa agents; . . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) **19545-26-7**, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab 154447-36-6, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylax
(delivery of therapeutic agents)

L7 ANSWER 9 OF 11 USPATFULL on STN

AB The present invention provides improved devices and methods for minimizing and/or inhibiting restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for programmed and controlled mizoribine delivery with increased efficacy to selected locations within a patient's vasculature to inhibit restenosis. An intraluminal delivery prosthesis may comprise an expansible structure and means on or within the structure for releasing mizoribine into the body lumen to inhibit smooth muscle cell proliferation.

DETD [0079] Mizoribine may be combined with other drugs (cytotoxic drugs,

cytostatic drugs, or **psoriasis** drugs, such as, mycophenolic acid, riboflavin, tiazofurin, methylprednisolone, FK 506, zafurin, methotrexate). One drug is in or coupled a first.

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) **19545-26-7**, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab 154447-36-6, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylax
(delivery of therapeutic agents)

L7 ANSWER 10 OF 11 USPATFULL on STN

AB The present invention provides improved devices and methods for minimizing and/or inhibiting restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for programmed and controlled methylprednisolone delivery with increased efficacy to selected locations within a patient's vasculature to inhibit restenosis. An intraluminal delivery prosthesis may comprise an expansible structure and means on or within the structure for releasing methylprednisolone into the body lumen to inhibit smooth muscle cell proliferation.

DETD [0079] Methylprednisolone may be combined with other drugs (cytotoxic drugs, cytostatic drugs, or **psoriasis** drugs, such as, mycophenolic acid, riboflavin, tiazofurin, mizoribine, FK 506, zafurin, methotrexate). For example, one drug is in or coupled.

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride

9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) **19545-26-7**, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab 154447-36-6, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylax
(delivery of therapeutic agents)

L7 ANSWER 11 OF 11 USPATFULL on STN

AB The invention provides pharmaceutical formulations and methods for the treatment of individuals suffering from a condition, disease, or disorder that is treatable by the inhibition of angiogenesis. The compositions comprise a Golgi apparatus disturbing agent in a substantially nontoxic amount effective to inhibit angiogenesis in a patient in need of anti-angiogenesis therapy, a solvent, and a pharmaceutically acceptable carrier. In preferred formulations, the Golgi apparatus disturbing agent is brefeldin A.

SUMM . . . overall health of an organism. For example, continuous or uncontrolled angiogenesis can cause or exacerbate diseases such as rheumatoid arthritis, **psoriasis**, and certain retinopathies, e.g., diabetic retinopathy. Furthermore, angiogenesis makes tumor growth and metastasis possible by vascularizing the tumor, thereby supplying.

SUMM . . . any patient who would benefit from inhibition of angiogenesis, the present method is particularly useful to treat individuals suffering from **psoriasis**, rheumatoid arthritis, retinopathy, and cellular proliferative diseases such as sarcomas, carcinomas, brain cancer, bladder cancer, breast cancer, colorectal cancer, head. . .

DETD . . . such as neoplasms, cancers, and tumors. "Cellular proliferative diseases" also include non-cancerous conditions such as benign melanomas, benign prostatic hyperplasia, **psoriasis**, and other cellular growths occurring within the epidermal layers.

DETD . . . or disorder that is treatable by at least partial inhibition of angiogenesis. Typically, patients suffering from arthritis, e.g., rheumatoid arthritis, **psoriasis**, or diabetic retinopathy, benefit from the present methods. Additionally, patients suffering from a neoplastic disease, i.e., a cellular proliferative disease, . . .

CLM What is claimed is:

. . . The method of claim 19, wherein the patient is suffering from a disease selected from the group consisting of arthritis, **psoriasis**, and diabetic retinopathy.

IT 50-18-0, Cyclophosphamide 50-44-2, Mercaptopurine 50-76-0, Actinomycin d 50-91-9, 5-Fluorodeoxyuridine 51-21-8, 5-Fu 51-75-2, Mechlorethamine 52-24-4, Thiotepa 53-19-0, Mitotane 55-98-1, Busulfan 56-53-1, Diethylstilbestrol 57-22-7, Vincristine 57-55-6, Propylene glycol, biological studies 58-05-9, Leucovorin 59-05-2, Methotrexate 60-29-7, Ethyl ether, biological studies 60-51-5, Phosphamide 64-17-5, Ethanol, biological studies 64-86-8, Colchicine 67-68-5, DmsO, biological studies 71-36-3, 1-Butanol, biological studies 76-43-7, Fluoxymesterone 77-76-9, 2,2-Dimethoxypropane 78-83-1, 2-Methyl-1-propanol, biological studies 78-92-2, 2-Butanol 78-93-3, Methyl ethyl ketone, biological studies 79-20-9, Methyl acetate 108-21-4, Isopropyl acetate 109-87-5, Dimethoxymethane 109-94-4, Ethyl formate 110-19-0, Isobutyl acetate 123-51-3, 3-Methyl-1-butanol 125-84-8, Aminogluthethimide 127-07-1, Hydroxyurea 127-19-5, Dimethylacetamide 127-31-1, Fludrocortisone 141-78-6, Ethyl acetate, biological studies 145-63-1, Suramin 147-94-4, Cytarabine 148-82-3, Melphalan 154-21-2, Lincomycin 154-42-7, 6-Thioguanine 154-93-8, Carmustine 305-03-3, Chlorambucil 320-67-2, 5-Azacytidine 446-72-0, Genistein 497-72-3, Methymycin 520-85-4, Medroxyprogesterone 564-25-0, Doxycycline 584-79-2, Bioallethrin 671-16-9, Procarbazine 865-21-4, Vinblastine 968-93-4, Testolactone 1404-00-8, Mitomycin 2098-66-0, Cyproterone 2998-57-4, Estramustine 3562-63-8, Megestrol 3778-73-2, Ifosfamide 4291-63-8, Cladribine 4342-03-4, Dacarbazine 9015-68-3, L-Asparaginase 10118-90-8, Minocycline 10540-29-1, Tamoxifen 11006-76-1, Streptogramin 11056-06-7, Bleomycin 11078-23-2, Copiamycin 12728-25-5, Desertomycin 12772-57-5, Radicicol 13010-47-4, Lomustine 13311-84-7, Flutamide 14769-73-4, Levamisole 15663-27-1, Cisplatin 18378-89-7, Mithramycin 18883-66-4, Streptozocin 19545-26-7, Wortmannin 19767-45-4, Mesna 20830-81-3, Daunorubicin 21679-14-1, Fludarabine 23214-92-8, Doxorubicin 29767-20-2, Teniposide 30562-34-6, Geldanamycin 33069-62-4, Paclitaxel 33419-42-0, Etoposide 41575-94-4, Carboplatin 50924-49-7, Bredinin 51264-14-3, Amsacrine 53123-88-9, Rapamycin 53643-48-4, Vindesine 53714-56-0, Leuprolide 53910-25-1, Deoxycoformycin 56420-45-2, Epirubicin 57982-77-1, Buserelin 57998-68-2, Aziridinybenzoquinone 58957-92-9, Idarubicin 62996-74-1, Staurosporine 63612-50-0, Nilutamide 65271-80-9, Mitoxantrone 65807-02-5, Goserelin 70288-86-7, Ivermectin 71486-22-1, Vinorelbine 72497-34-8, Sporaviridin 73989-17-0D, Avermectin, derivs. 83150-76-9, Octreotide 83314-01-6, Bryostatin 1 86090-08-6, Angiostatin 87806-31-3, Photofrin Porfimer sodium 90357-06-5, Bicalutamide 90996-54-6, Rhizoxin 95058-81-4, Gemcitabine 95152-88-8, Difficidin 95152-89-9, Oxydifficidin 97682-44-5, Irinotecan 109946-35-2, Tautomycin 110942-02-4, Aldesleukin 113507-06-5, Moxidectin 114977-28-5, Docetaxel 117704-25-3, Doramectin 120511-73-1, Anastrozole 123948-87-8, Topotecan 123997-26-2, Eprinomectin 127999-44-4, Tolytoxin 169181-40-2, Chivosazol a 187888-07-9, Endostatin 260362-86-5, Oocydin a (inhibitors of angiogenesis and tumor growth for local and systemic administration)

=> s 154447-36-6/RN

L9 39 154447-36-6/RN

=> S L9 and psoriasis

17597 PSORIASIS

L10 9 L9 AND PSORIASIS

=> d 110 1-9

L10 ANSWER 1 OF 9 USPATFULL on STN
AN 2003:273414 USPATFULL
TI Methods for modulating T cell responses by manipulating intracellular
signal transduction
IN June, Carl H., Rockville, MD, United States
PA The United States of America as represented by the Secretary of the
Navy, Washington, DC, United States (U.S. government)
PI US 6632789 B1 20031014
AI US 1994-245282 19940429 (8)
DT Utility
FS GRANTED
LN.CNT 1110
INCL INCLM: 514/001.000
INCLS: 514/453.000; 424/130.100; 424/278.100
NCL NCLM: 514/001.000
NCLS: 424/130.100; 424/278.100; 514/453.000
IC [7]
ICM: A01N061-00
ICS: A01N043-16; A61K039-395; A61K045-00
EXF 435/240.2; 435/240.1; 435/244; 424/278.1; 424/130.1; 514/453; 514/1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 2 OF 9 USPATFULL on STN
AN 2003:23354 USPATFULL
TI Intravascular delivery of mycophenolic acid
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PA Avantec Vascular Corporation, Sunnyvale, CA, UNITED STATES, 94086 (U.S.
corporation)
PI US 2003017190 A1 20030123
AI US 2002-242334 A1 20020911 (10)
RLI Division of Ser. No. US 2001-782927, filed on 13 Feb 2001, GRANTED, Pat.
No. US 6471980
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1014
INCL INCLM: 424/426.000
INCLS: 514/470.000; 514/171.000; 514/251.000; 514/291.000
NCL NCLM: 424/426.000
NCLS: 514/470.000; 514/171.000; 514/251.000; 514/291.000
IC [7]
ICM: A61K031-573
ICS: A61K031-525; A61K031-4745; A61F002-00; A61K031-365
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 9 USPATFULL on STN
AN 2002:259409 USPATFULL
TI Method for regulating angiogenesis
IN Hla, Timothy, Avon, CT, UNITED STATES
Lee, Meng-Jer, Unionville, CT, UNITED STATES
Claffey, Kevin P., Burlington, CT, UNITED STATES
Ancellin, Nicolas, Farmington, CT, UNITED STATES
Thangada, Shobha, Glastonbury, CT, UNITED STATES
PI US 2002142982 A1 20021003
AI US 2001-945353 A1 20010831 (9)
RLI Continuation-in-part of Ser. No. US 2000-651846, filed on 31 Aug 2000,
PENDING
PRAI US 1999-152266P 19990902 (60)
DT Utility
FS APPLICATION
LN.CNT 1830

INCL INCLM: 514/044.000
INCLS: 514/453.000
NCL NCLM: 514/044.000
NCLS: 514/453.000
IC [7]
ICM: A61K048-00
ICS: A61K031-366

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 4 OF 9 USPATFULL on STN
AN 2002:213450 USPATFULL
TI Intravascular delivery of mycophenolic acid
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PI US 2002114823 A1 20020822
US 6471980 B2 20021029
AI US 2001-782927 A1 20010213 (9)
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1135
INCL INCLM: 424/423.000
INCLS: 514/470.000
NCL NCLM: 424/423.000
NCLS: 424/424.000; 424/425.000; 424/426.000
IC [7]
ICM: A61F002-00
ICS: A61K031-365

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 9 USPATFULL on STN
AN 2002:158065 USPATFULL
TI Delivery or therapeutic capable agents
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PA AVANTEC VASCULAR CORPORATION, San Jose, CA (U.S. corporation)
PI US 2002082679 A1 20020627
AI US 2001-2595 A1 20011101 (10)
PRAI US 2000-258024P 20001222 (60)
US 2001-308381P 20010726 (60)
DT Utility
FS APPLICATION
LN.CNT 3153
INCL INCLM: 623/001.150
INCLS: 623/001.420; 424/426.000
NCL NCLM: 623/001.150
NCLS: 623/001.420; 424/426.000
IC [7]
ICM: A61F002-06
ICS: A61F002-00

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 9 USPATFULL on STN
AN 2002:158064 USPATFULL
TI Intravascular delivery of mizoribine
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PI US 2002082678 A1 20020627
AI US 2001-783254 A1 20010213 (9)
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION

LN.CNT 1050
INCL INCLM: 623/001.150
NCL NCLM: 623/001.150
IC [7]
ICM: A61F002-06
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 9 USPATFULL on STN
AN 2002:158063 USPATFULL
TI Intravascular delivery of methylprednisolone
IN Sirhan, Motasim, Sunnyvale, CA, UNITED STATES
Yan, John, Los Gatos, CA, UNITED STATES
PI US 2002082677 A1 20020627
AI US 2001-782804 A1 20010213 (9)
PRAI US 2000-258024P 20001222 (60)
DT Utility
FS APPLICATION
LN.CNT 1074

INCL INCLM: 623/001.150
INCLS: 623/001.430
NCL NCLM: 623/001.150
NCLS: 623/001.430
IC [7]
ICM: A61F002-06
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 8 OF 9 USPATFULL on STN
AN 2000:146409 USPATFULL
TI Use of rosmarinic acid and derivatives thereof as an immunosuppressant
or an inhibitor of SH2-mediated processes
IN Hur, Eun Mi, Kyonggi-do, Korea, Republic of
Choi, Young Bong, Kyonggi-do, Korea, Republic of
Park, Changwon, Kyonggi-do, Korea, Republic of
Lee, Jongsung, Seoul, Korea, Republic of
Park, Dongsu, Kyonggi-do, Korea, Republic of
Yun, Yungdae, Seoul, Korea, Republic of
Lee, Keun Hyeung, Seoul, Korea, Republic of
Oh, Jong-Eun, Seoul, Korea, Republic of
Ahn, Soon Choul, Taejon-si, Korea, Republic of
Lee, Hyun Sun, Taejon-si, Korea, Republic of
Ahn, Jong Sok, Taejon-si, Korea, Republic of
Jung, Soo Il, Kyonggi-do, Korea, Republic of
PA Mogam Biotechnology Research Institute, Korea, Republic of (non-U.S.
corporation)
PI US 6140363 20001031
AI US 1999-312405 19990514 (9)
PRAI KR 1998-17741 19980516
KR 1999-15989 19990504
DT Utility
FS Granted
LN.CNT 1179

INCL INCLM: 514/533.000
INCLS: 514/570.000
NCL NCLM: 514/533.000
NCLS: 514/570.000
IC [7]
ICM: A61K031-235
EXF 514/570; 514/533
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 9 OF 9 USPATFULL on STN
AN 2000:41069 USPATFULL

TI 12 (S)--hete receptor blockers
 IN Natarajan, Rama, Hacienda Heights, CA, United States
 Nadler, Jerry L., La Crescenta, CA, United States
 PA City of Hope, Duarte, CA, United States (U.S. corporation)
 PI US 6046224 20000404
 AI US 1998-172138 19981014 (9)
 PRAI US 1997-62335P 19971015 (60)
 DT Utility
 FS Granted
 LN.CNT 617
 INCL INCLM: 514/381.000
 INCLS: 514/560.000; 514/732.000; 424/254.100
 NCL NCLM: 514/381.000
 NCLS: 424/254.100; 514/560.000; 514/732.000
 IC [7]
 ICM: A61K031-41
 EXF 424/254.1; 514/381; 514/732; 514/560
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d 110 ab, kwic

L10 ANSWER 1 OF 9 USPATFULL on STN

AB Methods for modulating T cell responses by manipulating intracellular signals associated with T cell costimulation are disclosed. The methods involve inhibiting or stimulating the production of at least one D3-phosphoinositide in a T cell. Production of D3-phosphoinositides can be manipulated by contacting a T cell with an inhibitor or activator of phosphatidylinositol 3-kinase. Inhibitors of phosphatidylinositol 3-kinase for use in the methods of the invention include wortmannin and quercetin, or derivatives or analogues thereof. The methods of the invention can further comprise modulating other intracellular signals associated with costimulation, such as protein tyrosine phosphorylation, for example by modulating the activity of a protein tyrosine kinase or a protein tyrosine phosphatase in the T cell. Inhibition of a T cell response in accordance with the disclosed methods is useful therapeutically in situations where it is desirable to inhibit an immune response to an antigen(s), for example in organ or bone marrow transplantation and autoimmune diseases. Alternatively, stimulation of a T cell response in accordance with the disclosed methods is useful therapeutically to enhance an immune response to an antigen(s), for example to stimulate an anti-tumor response in a subject with a tumor, to stimulate a response against a pathogenic agent or increase the efficacy of vaccination. Novel screening assays for identifying inhibitors or activators of phosphatidylinositol 3-kinase, which can be used to inhibit or stimulate a T cell response, are also disclosed.

DETD . . . disorders associated with an inappropriate or abnormal immune response include rheumatoid arthritis, juvenile rheumatoid arthritis, psoriatic arthritis, allergies, contact dermatitis, **psoriasis**, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, multiple sclerosis, allergic encephalomyelitis, systemic lupus erythematosus, acute necrotizing hemorrhagic encephalopathy, idiopathic.

IT 117-39-5, Quercetin 19545-26-7, Wortmannin 70563-58-5, Herbimycin A **154447-36-6**, LY 294002
 (methods for modulating T-cell responses by manipulating intracellular signal transduction)

=> d 110 ab, kwic 2-9

L10 ANSWER 2 OF 9 USPATFULL on STN

AB The present invention provides improved devices and methods for minimizing and/or inhibiting restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for programmed and controlled mycophenolic acid delivery with increased efficacy to selected locations within a patient's vasculature to inhibit restenosis. An intraluminal delivery prosthesis may comprise an expansible structure and means on or within the structure for releasing mycophenolic acid at a rate selected to inhibit smooth muscle cell proliferation.

DETD [0084] MPA may be combined with other drugs (cytotoxic drugs, cytostatic drugs, or **psoriasis** drugs, such as, mizoribine, riboflavin, tiazofurin, methylprednisolone, FK 506, zafurin, methotrexate). One drug is in or coupled a first coat. . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) 19545-26-7, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab **154447-36-6**, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylax (delivery of therapeutic agents)

L10 ANSWER 3 OF 9 USPATFULL on STN

AB Methods for the inhibition of angiogenesis are presented, comprising affecting the response of the EDG-1 receptor by the administration of pharmaceutically effective antagonists of EDG-1 signal transduction. This invention is based in part on the discovery that Akt protein kinase phosphorylation is required for endothelial cell chemotaxis mediated by the EDG-1 G protein-coupled receptor. This invention relates to the use of modifiers of EDG-1 signal transduction to treat disorders of undesired angiogenesis.

SUMM . . . as solid tumor growth, heart disease, rheumatoid arthritis, peripheral vascular diseases of the elderly, diabetic retinopathy, Kaposi's sarcoma, hemangioma, and **psoriasis**. Angiogenesis is

prominent in solid tumor formation and metastasis. Angiogenic factors have been found associated with several solid tumors. A. . . .

SUMM . . . of EDG-1 signal transduction is used to treat undesired angiogenesis in tumors, rheumatoid arthritis, diabetic retinopathy, Kaposi's sarcoma, hemangioma or **psoriasis**.

DETD . . . in the neovascularization of tumor cells or other pathological conditions such as rheumatoid arthritis, diabetic retinopathy, Kaposi's sarcoma, hemangioma, and/or **psoriasis**. The oligonucleotides may be adapted or formulated for administration to the body in a number of ways suitable for the. . . .

IT 19545-26-7, Wortmannin 26993-30-6 **154447-36-6**, LY294002 (EDG-1 signal transduction modifiers for regulating angiogenesis)

L10 ANSWER 4 OF 9 USPATFULL on STN

AB The present invention provides improved devices and methods for minimizing and/or inhibiting restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for programmed and controlled mycophenolic acid delivery with increased efficacy to selected locations within a patient's vasculature to inhibit restenosis. An intraluminal delivery prosthesis may comprise an expansible structure and means on or within the structure for releasing mycophenolic acid at a rate selected to inhibit smooth muscle cell proliferation.

DETD [0084] MPA may be combined with other drugs (cytotoxix drugs, cytostatic drugs, or **psoriasis** drugs, such as, mizoribine, riboflavin, tiazofurin, methylprednisolone, FK 506, zafurin, methotrexate). One drug is in or coupled a first coat. . . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyrindamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) 19545-26-7, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5, Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid) 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid) 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1, Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3, Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8, Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6, Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil 143653-53-6, Rheopro 152923-56-3, Daclizumab **154447-36-6**, LY 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7, Eptifibatide 439112-98-8, Parylast

(delivery of therapeutic agents)

L10 ANSWER 5 OF 9 USPATFULL on STN

AB A device and a method using the same, for reducing restenosis and hyperplasia after intravascular intervention. In particular, the present invention provides luminal prostheses which allow for controlled release of at least one therapeutic capable agent with increased efficacy to selected locations within a patient's vasculature to reduce restenosis. An intraluminal prosthesis may comprise an expandable structure and a source adjacent the expandable structure for releasing the therapeutic capable agent into the body lumen to reduce smooth muscle cell proliferation.

SUMM . . . anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs such as Thalidomide.TM.; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; riboflavin; tiazofurin; zafurin; anti-platelet agents including cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors such as clopidogrel (e.g., Plavix.TM.). . .

DETD . . . amethopterin, is an immunosuppressant and anti-proliferative agent that has been used in the treatment of certain neoplastic diseases and severe **psoriasis**. Chemically Methotrexate.TM. is N-[4[[(2,4-diamino-6-pteridinyl)methyl] methylamino]benzoyl]-L-glutamic acid. In particular, Methotrexate.TM. is a is inhibits dihydrofolic acid reductase, thereby inhibiting the reduction. . .

DETD . . . anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs such as Thalidomide.TM.; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; riboflavin; tiazofurin; zafurin; anti-platelet agents including cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors such as clopidogrel (e.g., Plavix.TM.). . .

DETD [0180] The therapeutic capable agent may be combined with a second therapeutic capable agent (cytotoxic drugs, cytostatic drugs, or **psoriasis** drugs). One agent is in or coupled to a first coat while other agent is in or coupled to a . . .

CLM What is claimed is:

. . . agents; radiolabelled agents; anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; anti-platelet agents including, cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors ticlopidipine phosphodiesterase III inhibitors, glycoprotein IIb/IIIa agents; eptifibatides, . . .

. . . agents; radiolabelled agents; anti-coagulants such as heparin and its derivatives; anti-angiogenesis drugs; angiogenesis drugs; PDGF-B and/or EGF inhibitors; anti-inflammatories including **psoriasis** drugs; anti-platelet agents including, cyclooxygenase inhibitors such as acetylsalicylic acid, ADP inhibitors ticlopidipine phosphodiesterase III inhibitors, glycoprotein IIb/IIIa agents; . . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2, Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone, biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6, Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8, Cellulose acetate butyrate 9005-49-6, Heparin, biological studies 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate) 9016-00-6, Poly(dimethyl siloxane) 19545-26-7, Wortmannin 24280-93-1, Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol

copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5,
 Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol
 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate
 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid)
 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid)
 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1,
 Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol
 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine
 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3,
 Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic
 acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU
 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer
 83120-66-5, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus
 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8,
 Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6,
 Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil
 143653-53-6, Rheopro 152923-56-3, Daclizumab **154447-36-6**, LY
 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7,
 Eptifibatide 439112-98-8, Parylant
 (delivery of therapeutic agents)

L10 ANSWER 6 OF 9 USPTFULL on STN

AB The present invention provides improved devices and methods for
 minimizing and/or inhibiting restenosis and hyperplasia after
 intravascular intervention. In particular, the present invention
 provides luminal prostheses which allow for programmed and controlled
 mizoribine delivery with increased efficacy to selected locations within
 a patient's vasculature to inhibit restenosis. An intraluminal delivery
 prosthesis may comprise an expansible structure and means on or within
 the structure for releasing mizoribine into the body lumen to inhibit
 smooth muscle cell proliferation.

DETD [0079] Mizoribine may be combined with other drugs (cytotoxix drugs,
 cytostatic drugs, or **psoriasis** drugs, such as, mycophenolic
 acid, riboflavin, tiazofurin, methylprednisolone, FK 506, zafurin,
 methotrexate). One drug is in or coupled a first. . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide
 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2,
 Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone
 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone,
 biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic
 anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6,
 Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol
 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride
 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl
 methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8,
 Cellulose acetate butyrate 9005-49-6, Heparin, biological studies
 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate)
 9016-00-6, Poly(dimethyl siloxane) 19545-26-7, Wortmannin 24280-93-1,
 Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer
 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol
 copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5,
 Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol
 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate
 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid)
 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid)
 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1,
 Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol
 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine
 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3,
 Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic
 acid copolymer 67291-18-3, Poly(3-hydroxyvaleric acid), SRU

73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer
 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin
 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus
 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8,
 Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6,
 Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil
 143653-53-6, Rheopro 152923-56-3, Daclizumab **154447-36-6**, LY
 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7,
 Eptifibatide 439112-98-8, Parylast
 (delivery of therapeutic agents)

L10 ANSWER 7 OF 9 USPTFULL on STN

AB The present invention provides improved devices and methods for
 minimizing and/or inhibiting restenosis and hyperplasia after
 intravascular intervention. In particular, the present invention
 provides luminal prostheses which allow for programmed and controlled
 methylprednisolone delivery with increased efficacy to selected
 locations within a patient's vasculature to inhibit restenosis. An
 intraluminal delivery prosthesis may comprise an expansible structure
 and means on or within the structure for releasing methylprednisolone
 into the body lumen to inhibit smooth muscle cell proliferation.

DETD [0079] Methylprednisolone may be combined with other drugs (cytotoxix
 drugs, cytostatic drugs, or **psoriasis** drugs, such as,
 mycophenolic acid, riboflavin, tiazofurin, mizoribine, FK 506, zafurin,
 methotrexate). For example, one drug is in or coupled. . .

IT 50-02-2, Dexamethasone 50-18-0, Cyclophosphamide 50-35-1, Thalidomide
 50-78-2, Acetylsalicylic acid 53-03-2, Prednisone 58-32-2,
 Dipyridamole 59-05-2, Methotrexate 83-43-2, Methylprednisolone
 83-88-5, Riboflavin, biological studies 88-12-0, N-Vinyl-2-pyrrolidone,
 biological studies 107-73-3, Phosphorylcholine 108-31-6, Maleic
 anhydride, biological studies 127-07-1, Hydroxyurea 446-86-6,
 Azathioprine 1402-38-6, Actinomycin 1972-08-3, Dronabinol
 7689-03-4, Camptothecin 9002-84-0 9002-86-2, Polyvinyl chloride
 9002-98-6 9003-05-8, Polyacrylamide 9003-63-8, Poly(n-butyl
 methacrylate) 9004-34-6, Cellulose, biological studies 9004-36-8,
 Cellulose acetate butyrate 9005-49-6, Heparin, biological studies
 9007-27-6, Chondroitin 9011-14-7, Poly(methyl methacrylate)
 9016-00-6, Poly(dimethyl siloxane) 19545-26-7, Wortmannin 24280-93-1,
 Mycophenolic acid 24937-78-8, Ethylene-vinyl acetate copolymer
 24980-41-4, Polycaprolactone 25067-34-9, Ethylene-vinyl alcohol
 copolymer 25189-52-0 25248-42-4, Polycaprolactone 25249-16-5,
 Poly(2-hydroxyethyl methacrylate) 25322-68-3, Polyethylene glycol
 25722-33-2, Parylene 25736-86-1, Polyethylene glycol methacrylate
 26009-03-0, Poly(glycolic acid) 26023-30-3, Poly(lactic acid)
 26063-00-3, Poly(hydroxybutyrate) 26100-51-6, Poly(lactic acid)
 26124-68-5, Poly(glycolic acid) 26744-04-7 29223-92-5 31621-87-1,
 Polydioxanone 31900-57-9, Poly(dimethyl siloxane) 33069-62-4, Taxol
 35284-36-7 38748-32-2, Triptolide 50924-49-7, Mizoribine
 53123-88-9, Rapamycin 55142-85-3, Ticlopidine 59865-13-3,
 Cyclosporine 60084-10-8, Tiazofurin 66844-36-8, Caprolactone-L-lactic
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 73963-72-1, Pletal 80137-67-3, Caprolactone-lactic acid copolymer
 83120-66-5, Poly(3-hydroxyvaleric acid) 89149-10-0, Deoxyspergualin
 95058-81-4, Gemcitabine 99614-02-5, Zofran 104987-11-3, Tacrolimus
 104987-12-4, Ascomycin 105979-17-7, Benidipine 107007-99-8,
 Granisetron hydrochloride 113665-84-2, Clopidogrel 120202-66-6,
 Plavix 123948-87-8, Topotecan 128794-94-5, Mycophenolate mofetil
 143653-53-6, Rheopro 152923-56-3, Daclizumab **154447-36-6**, LY
 294002 159351-69-6, Certican 162359-56-0, FTY 720 188627-80-7,
 Eptifibatide 439112-98-8, Parylast
 (delivery of therapeutic agents)

L10 ANSWER 8 OF 9 USPATFULL on STN

AB The present invention relates to use of rosmarinic acid and/or derivatives thereof as immunosuppressive agents and/or as inhibitor of SH2 domain function. Disclosed in the present invention is that rosmarinic acid and derivatives thereof specifically inhibit the binding of ligand peptides to Lck SH2 domain, disturb the Lck-mediated signal transduction in T cells, also inhibit cytoline gene expression, and suppress immune responses in the transplanted tissue. These activities of rosmarinic acid and derivatives thereof support their applicability to treatment, prevention and/or diagnosis of graft rejection, GVHD, autoimmune diseases, inflammatory diseases, etc.

DETD . . . liver, bone marrow and skin transplants; autoimmune diseases such as lupus, rheumatoid arthritis, diabetes mellitus, myasthenia gravis, multiple sclerosis and **psoriasis**; diseases of inflammation such as dermatitis, eczema, seborrhea and inflammatory bowel disease; and fungal infections.

DETD . . . transplantation rejection, autoimmune disease and inflammatory disease, more specifically of GVHD, lupus, rheumatoid arthritis, diabetes mellitus, myasthenia gravis, multiple sclerosis, **psoriasis**, dermatitis, eczema, seborrhea, inflammatory bowel disease, Crohn's disease, primary biliary cirrhosis, etc.

DETD . . . liver, bone marrow and skin transplants; autoimmune diseases such as lupus, rheumatoid arthritis, diabetes mellitus, myasthenia gravis, multiple sclerosis and **psoriasis**; diseases of inflammation such as dermatitis, eczema, seborrhea and inflammatory bowel disease; and fungal infections.

CLM What is claimed is:

. . . The method of claim 1, wherein the autoimmune disease includes lupus, rheumatoid arthritis, diabetes mellitus, myasthenia gravis, multiple sclerosis or **psoriasis**.

. . . The method of claim 15, wherein the autoimmune disease includes lupus, rheumatoid arthritis, diabetes mellitus, myasthenia gravis, multiple sclerosis or **psoriasis**.

IT 154447-36-6, LY294002

(rosmarinic acid and derivs. as immunosuppressants and inhibitors of SH2-mediated processes)

L10 ANSWER 9 OF 9 USPATFULL on STN

AB The 12-lipoxygenase product, 12(S)-HETE, mediates hyperproliferative and hyperplastic responses seen in atherosclerosis, diabetes, Parkinson's disease, Alzheimer's, stroke-induced nerve damage and cancer. 12-HETE also mediates inflammation and cell death in some cell systems, particularly B-islet cells of the pancreas. The present invention involves amelioration of disease states mediated by 12(S)-HETE by blocking specific 12(S)-HETE receptors.

SUMM . . . receptor. 12(S)-HETE, a product of the 12-lipoxygenase pathway, mediates the hyperproliferative and inflammatory responses present in such diseases as atherosclerosis, **psoriasis**, diabetes, and cancer. 12(S)-HETE also mediates inflammatory responses and cell death in some cell types, particularly pancreatic islet beta cells. . .

IT 11128-99-7, Angiotensin II 154447-36-6, LY294002

(HETE receptor blockers, and therapeutic use)

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(FILE 'HOME' ENTERED AT 11:28:33 ON 01 MAR 2004)

FILE 'REGISTRY' ENTERED AT 11:28:42 ON 01 MAR 2004

L1 0 S WORMANNIN/CN

L2 1 S WORTMANNIN/CN
L3 0 S LY294002/CN
L4 1 S LY 294002/CN

FILE 'USPATFULL' ENTERED AT 11:31:10 ON 01 MAR 2004

L5 0 S 19545-26-7 RN
L6 54 S 19545-26-7/RN
L7 11 S L6 AND PSORIASIS
L8 0 S L7 AND PD<1999
L9 39 S 154447-36-6/RN
L10 9 S L9 AND PSORIASIS

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